# SEPARATE SENSORY CENTERS IN THE PARIETAL LOBE FOR THE LIMBS.[1]

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Friedrich Müller has recently made a careful study of the disturbances of sensation resulting from lesions of the brain. He mentions that cerebral hemianesthesia without motor paralysis is seldom seen, if hysterical hemianesthesia be excluded, and that many authors deny the possibility of such an occurrence. Dejerine, among others, has said that cerebral hemianesthesia is always associated with hemiplegia, and that the most intense sensory alterations are found in the weaker limb, although no parallelism exists between the degree of the hemiplegia and that of the hemianesthesia.

Munk, Tripier, Dejerine, Henschen and others hold that the motor cortical areas are also sensory, and Dejerine says he has never seen a lesion in the region of the supramarginal gyrus which caused sensory disturbances without motor disturbances. One must acknowledge, according to Müller, that cortical sensory disturbances seldom occur without weakness of the limbs in which sensation is disturbed.

Von Monakow has accepted the view that the motor area is confined to the precentral convolution, and that the sensory region is in the postcentral convolution and parietal lobe; and he refers in support of this view to cases of cortical hemiplegia without hemianesthesia and to cases of cortical hemianesthesia without hemiplegia. The experience of surgeons seems to show that the precentral convolution is not an essential part of the sensory area, although it cannot be denied that a lesion of this convolution may occasionally cause disturbance of stereognosis. Von Monakow holds, from a study of secondary degeneration, that the pyramidal tract arises only in the precentral convolution.

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[1] Read before the Philadelphia Neurological Society, Dec. 19, 1905. From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania, and from the Polyclinic Hospital.

This is the presentation of the subject as given by Müller, and to the cases of hemianesthesia without hemiplegia from lesions of the parietal lobe mentioned by v. Monakow, (cases of Cox, Mills, Redlich, Spiller, Oppenheim, Durante, v. Monakow), Müller adds the cases of Prevost, Wilbrand, Lang, Henschen (4), Jany, Long, and five cases of his own. He shows that subjective disturbances of sensation (paresthesia) may be more extensive than objective sensory disturbances from a cortical lesion.[2]

Among American writers the one who has longest and most persistently maintained the separation of the motor and sensory areas of the brain is C. K. Mills. At the meeting of the American Neurological Association in St. Louis in Sept. 1904, he suggested that the cortical sensory area for the upper limb is probably separate from that for the lower limb. This view has been entertained by others also, thus Alfred W. Campbell[3] says: "I incline to the belief that the area for common sensation, like the motor area, will ultimately be divided into different levels or centers." Doubtless many others have held a similar opinion, but there has been little real foundation for it. In the same paper Campbell remarks: "Proof, from the pathological side, of the correctness of this localisation [of the motor area anterior to the central fissure] has been supplied by an examination of the brains of cases of amyotrophic lateral sclerosis. Amyotrophic lateral sclerosis is a perfect example of a malady limited in its affection to the muscles and the motor system of neurones; and on making serial sections of the central gyri in two typical instances, it was extremely instructive to find profound changes confined absolutely to our 'precentral or motor area' and consisting essentially of a destruction of the cells of Betz."

The idea of employing cases of amyotrophic lateral sclerosis to limit the motor area, was suggested by me[4] in 1900, and in my report of a case of this disease studied with this object of localization in view by the Marchi method, mention is made of degenerated fibers in the postcentral convolution. They were fewer by far than were the degenerated fibers in the precentral convolution, but they were unquestionably present. I have recently re-examined these sections. It is true that the relative intensity of the degeneration in the postcentral and precentral convolutions, as represented by the artist in the diagrammatic drawing accompanying my paper, is not correct. The difference in the results reached by Campbell and by me may depend on the fact that he relied on alteration of the cells of Betz, and I on degeneration of nerve fibers as shown by the Marchi method.

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[2] Friedrich Müller, Sammlung Klinischer Vorträge, Innere Medizin, No. 118-19, 1905.

[3] Campbell. The Journal of Mental Science, Jan. 1904, p. 651.

I have had recently an opportunity to study a case which shows that sensation may be affected from a cerebral cortical lesion, without motor paralysis; but still more important, that the sensory alteration may be confined to one limb. From this we may conclude that the sensory centers for the limbs are distinct from one another.

M. W., male, 38 years of age, teacher, came to my clinic at the Polyclinic Hospital, in Aug. or Sept., 1905.

He was perfectly well until July 21, 1904. He was struck on that day over the right parietal lobe with a club by a man who intended to injure him. He was unconscious one or two hours and was confined to his bed about ten days after he received the blow. He was unable to move the fingers of his left hand during the two weeks following the injury and could not make a fist. It is impossible to say whether this inability was the result of motor weakness or of sensory disturbances. He could move his lower limb the day following the injury. He is uncertain as to whether the lower part of his face was paretic or not. The hand at first felt "dead," and he has had paresthesia in it since the injury. He has never had any subjective or objective disturbances of sensation in the left lower limb or face.

Present condition, Dec. 11, 1905: The movements of the left upper limb are awkward, although the limb can be moved freely at all parts, and is not weak. He is unable to button his coat with his left hand alone when his eyes are closed, because of incoordination of the fingers, but can button it promptly with his right hand. The sense of position is greatly impaired in the fingers of his left hand, and he is usually unable to tell which finger is moved, or when a finger is bent backward or forward. Stereognostic perception is also greatly impaired in his left hand, and usually he is unable to name any object placed in this hand if he does not see it. Occasionally if the object falls from the hand he is unaware of the loss. Sensations of touch, pain and temperature are diminished in the left hand, but not to the same degree as are the sense of position and stereognostic perception. He will occasionally answer correctly when his left hand is touched or pricked with a pin, but usually his answers are uncertain and in marked contrast to the positiveness with which he replies to sensory tests of the right hand. The sensation of touch is a little less acute in the left hand than in the left arm above the elbow. Pain sensation is less altered than tactile sensation, and temperature sensations for both cold and warmth are more nearly equal in the two upper extremities. The spacing sensation is also greatly altered in the left hand, and he cannot tell whether the thumb and first finger are near together or far apart. In the right hand he can tell whether the thumb and first finger are one or two inches apart. Sensations on the chest seem to be the same on the two sides.

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[4] Spiller. THE JOURNAL OF NERVOUS AND MENTAL DISEASE, 1900, p. 165, and Contributions from the Wm. Pepper Laboratory of Clinical Medicine, vol. 1, 1900.

The left lower limb is not affected. His answers to sensory stimuli are always correct and prompt. He recognizes at once any movement of the toes and the direction in which they are moved. The limb is not weak. The patellar reflexes are not prompt. Sensation in the left side of the face appears to be entirely normal, and the right side of the body is not affected. Hemianopsia is not present.

Extremely interesting in this case are the marked sensory alterations of the left upper limb, with no disturbances of the left side of the face and of the left lower limb, and no motor palsy of the left limbs, at least at the present time; the symptoms resulting from a blow given over the right parietal lobe. The case seems to indicate that the sensory center for the upper limb must be distinct from those for the face and lower limb. I am inclined to place the lesion in the lower part of the parietal lobe, in part because this area is near the motor center for the upper limb, and in part because of a case I[5] reported in 1898. This was as follows: A man complained, during the night preceding his attack, of fatigue, headache and inability to sleep. The following morning he fell to the floor on attempting to arise from his bed, but was not unconscious. When he was found he had paresis of the left limbs and of the left side of the face and tongue. Later the patient could raise his left upper limb above his head, but every movement of this limb was ataxic in an extreme degree, though the limb was not paralyzed. The mental condition of the patient prevented an examination of the condition of sensation. At the necropsy a hemorrhage was found in the right parietal lobe. The hemorrhagic area was about 2 centimetres in diameter, and extended inward in the form of a cone to the lateral ventricle, having its base in the cortex. It was situated about 4 centimetres from the longitudinal fissure, and about 2 centimetres or 3 centimetres behind the Rolandic fissure. The injury was in, or very close to, the supramarginal gyrus. The ataxia was probably caused by a loss of the sense of position.

The case that I now report seems to indicate that the alterations of the sensations of position and space and of the stereognostic perception is greater from a lesion of the parietal lobe than is the alteration of the sensations of touch, pain and temperature.

In a paper published in 1904 I[6] discussed the location of certain forms of sensation in the parietal lobe, and reported two cases with necropsy. In one of these the loss of stereognostic perception and of the sense of position in the left hand, the awkward movements of the left fingers, and the impairment of sensation for touch, but not pain, in the left upper limb were attributable to tuberculous plaques almost confined to the right parietal lobe. I could find no case of this tuberculous meningitis in plaques described in American literature, and few in foreign. The other case was one in which ataxia, paresis and astereognosis of the left upper limb were caused by softening of the right parietal lobe.

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[5] Spiller. JOURNAL OF NERVOUS AND MENTAL DISEASE, Jan., 1899, p. 43.

[6] Spiller. "A Report of Five Cases of Tumor of the Brain with Necropsy." The American Journal of the Medical Sciences, Feb., 1904, p. 311.